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(FILE 'HOME' ENTERED AT 08:59:11 ON 28 JAN 2002)

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CABA,
CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,
DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 08:59:22 ON
28 JAN 2002

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L1 QUE (PROTEIN C)

FILE 'MEDLINE, CANCERLIT, SCISEARCH, EMBASE, BIOSIS' ENTERED AT 09:04:57
 ON 28 JAN 2002

L2 12887 S L1 AND (THROMBOCYTOPENIC (W) PURPURA) OR (HEMOLYTIC
 (W) UREM?)
 L3 2881 S L2 AND TREAT?
 L4 1526 DUP REM L3 (1355 DUPLICATES REMOVED)
 L5 90408 S PROTEIN (W) C
 L6 12846 S L5 (S) (THROMBOCYTOPENIC PURPURA) OR (HEMOLYTIC UREMI?)
 L7 16 S L4 AND (PROTEIN C)
 L8 16 DUP REM L7 (0 DUPLICATES REMOVED)

=> d 18 ibib ab 1-16

L8 ANSWER 1 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001127661 EMBASE
TITLE: Review of management of purpura fulminans and two case reports.
AUTHOR: Nolan J.; Sinclair R.
CORPORATE SOURCE: J. Nolan, Department of Anaesthesia, Bristol Royal Infirmary, Marlborough Street, Bristol BS2 8HW, United Kingdom
SOURCE: British Journal of Anaesthesia, (2001) 86/4 (581-586).
Refs: 26
ISSN: 0007-0912 CODEN: BJANAD
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
024 Anesthesiology
025 Hematology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Purpura fulminans (PF) is a haemorrhagic condition usually associated with

sepsis or previous infection. Features include tissue necrosis, small vessel thrombosis and disseminated intravascular coagulation. Gram-negative organisms are the commonest cause of the acute infectious type, which is often associated with multi-organ failure. An idiopathic variety, however, is often confined to the skin. The mortality rate has decreased with better **treatment** of secondary infections, supportive care and new **treatments**, but it remains a disabling condition often requiring major amputations. We describe two cases and review the various **treatments** for this condition.

L8 ANSWER 2 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001442143 EMBASE
TITLE: Advances in the understanding of the pathogenetic pathways of disseminated intravascular coagulation result in more insight in the clinical picture and better management strategies.
AUTHOR: Levi M.; De Jonge E.; Van der Poll T.; Ten Cate H.
CORPORATE SOURCE: Dr. M. Levi, Dept. of Vascular Medicine, Academic Medical Center, University of Amsterdam, Meibergdreef 9, 1105 AZ Amsterdam, Netherlands. m.m.levi@amc.uva.nl
SOURCE: Seminars in Thrombosis and Hemostasis, (2001) 27/6 (569-575).
Refs: 75
ISSN: 0094-6176 CODEN: STHMBV
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 025 Hematology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Disseminated intravascular coagulation (DIC) is a syndrome characterized by systemic intravascular activation of coagulation leading to widespread deposition of fibrin in the circulation. There is ample experimental and pathological evidence that the fibrin deposition contributes to multiple organ failure. The massive and ongoing activation of coagulation may

Post dated

result in depletion of platelets and coagulation factors, which may cause bleeding (consumption coagulopathy). Recent knowledge on important pathogenetic mechanisms that may lead to DIC has resulted in novel preventive and therapeutic approaches to patients with DIC. DIC is not a disease in itself but is a complication of a variety of disorders. However, the pathogenesis of DIC follows similar pathways in almost all

of

these situations, with a pivotal role of proinflammatory cytokines. The cornerstone of the management of DIC is the specific and vigorous **treatment** of the underlying disorder. Strategies aimed at the inhibition of coagulation activation may theoretically be justified and have been found to be beneficial in experimental and initial clinical studies. These strategies comprise inhibition of tissue factor-mediated activation of coagulation and restoration of physiological anticoagulant pathways by means of the administration of (activated) **protein C** concentrate or antithrombin concentrate.

L8 ANSWER 3 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001169016 EMBASE
TITLE: Evidence-based **treatment** of patients with ischemic cerebrovascular disease.
AUTHOR: Llinas R.; Caplan L.R.
CORPORATE SOURCE: Dr. R. Llinas, Department of Neurology, Johns Hopkins - Bayview Med. Center, B122b 4940 Eastern Avenue, Baltimore, MD 21224, United States. rllinas@jhmi.edu
SOURCE: Neurologic Clinics, (2001) 19/1 (79-105).
Refs: 142
ISSN: 0733-8619 CODEN: NECLEG
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Primary and secondary prevention of stroke has changed considerably during the last ten years. Numerous trial have studied the use and efficacy of various **treatment** that were previously used and defended on theoretical or anecdotal grounds. This article discusses cerebrovascular disease in subsets and reviews **treatments** based on evidence in each case.

L8 ANSWER 4 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001014190 EMBASE
TITLE: Blood components for hemostasis.
AUTHOR: Teruya J.; Ramsey G.
CORPORATE SOURCE: Dr. J. Teruya, Department of Pathology, Northwestern University Med. Sch., Chicago, IL, United States
SOURCE: Laboratory Medicine, (2001) 32/1 (31-35).
Refs: 7
ISSN: 0007-5027 CODEN: LBMEBX
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 025 Hematology
037 Drug Literature Index
LANGUAGE: English

L8 ANSWER 5 OF 16 MEDLINE
ACCESSION NUMBER: 2001231953 MEDLINE
DOCUMENT NUMBER: 21030993 PubMed ID: 11190905
TITLE: Increased plasma thrombomodulin as a vascular endothelial cell marker in patients with thrombotic **thrombocytopenic purpura** and **hemolytic uremic syndrome**.

AUTHOR: Mori Y; Wada H; Okugawa Y; Tamaki S; Nakasaki T; Watanabe R; bazza E C; Nishikawa M; Minami Shiku H
CORPORATE SOURCE: Misaki Red Cross Blood Center, Mie University School of Medicine, Tsu-city, Japan.
SOURCE: CLINICAL AND APPLIED THROMBOSIS/HEMOSTASIS, (2001 Jan) 7 (1) 5-9.
JOURNAL code: DAV; 9508125. ISSN: 1076-0296. *Post dated*
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010503

AB Several hemostatic and vascular endothelial cell markers were measured in 39 patients with thrombotic **thrombocytopenic purpura** (TTP)/**hemolytic uremic syndrome** (HUS) and in 20 healthy volunteers to examine the relationship between the occurrence of hemostatic abnormality or vascular endothelial cell injury and patient outcome. The plasma levels of von Willebrand factor, tissue plasminogen activator (TPA), plasminogen activator inhibitor (PAI-1), and the TPA-PAI-1 complex were significantly increased in TTP/HUS patients; however, the levels of these markers were not significantly different between TTP/HUS patients who survived and those who died, suggesting that these markers might not be directly related to outcome. The plasma levels of soluble granule membrane protein (GMP)-140 were significantly higher in TTP/HUS patients than in healthy volunteers, suggesting that platelets and vascular endothelial cells are activated or injured in TTP/HUS. There was no significant difference in GMP-140 levels between TTP/HUS patients with good and poor prognoses; this may be owing to the release of GMP-140 from platelets. The plasma thrombomodulin (TM) levels in TTP/HUS patients were significantly higher than in healthy volunteers; the plasma TM levels were significantly higher in patients who died than in patients who survived. These findings showed that TM levels reflect the outcome and that the outcome of TTP/HUS depends on the presence vascular endothelial cell injury. The plasma **protein C** and antithrombin levels were markedly reduced in TTP/HUS patients who died compared with those who survived. These findings suggest that reduced plasma antithrombin and **protein C** may be useful markers of systemic vascular endothelial injury. In conclusion, the results of this study showed that the outcome of TTP/HUS is related to vascular endothelial cell injury and that plasma TM, antithrombin, and **protein C** levels may be useful markers of systemic vascular endothelial cell injury.

L8 ANSWER 6 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000318539 EMBASE
TITLE: Peri-operative management of patients with coagulation disorders.
AUTHOR: Martlew V.J.
CORPORATE SOURCE: V.J. Martlew, Department of Haematology, Royal Liverpool University Hospital, Prescott Street, Liverpool L7 8XP, United Kingdom
SOURCE: British Journal of Anaesthesia, (2000) 85/3 (446-455).
Refs: 52
ISSN: 0007-0912 CODEN: BJANAD
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 009 Surgery
024 Anesthesiology
025 Hematology
037 Drug Literature Index

LANGUAGE: English

L8 ANSWER 7 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCIENCE B.V.
ACCESSION NUMBER: 2000300403 EMBASE
TITLE: Current management of disseminated intravascular coagulation.
AUTHOR: Levi M.; De Jonge E.
SOURCE: Hospital Practice, (15 Aug 2000) 35/8 (59-66).
ISSN: 8750-2836 CODEN: HOPRBW
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 006 Internal Medicine
009 Surgery
010 Obstetrics and Gynecology
025 Hematology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Both a bleeding and a thrombotic disorder, disseminated intravascular coagulation presents diagnostic and therapeutic challenges. At present, diagnosis requires a set of blood tests; therapy focuses on reversing the underlying disorder and providing supportive **treatment**. Clinical studies of specific tests and **treatments** are now under way.

L8 ANSWER 8 OF 16 MEDLINE

ACCESSION NUMBER: 2000423782 MEDLINE
DOCUMENT NUMBER: 20395811 PubMed ID: 10936861
TITLE: Plasma levels of activated **protein C**-**protein C** inhibitor complex in patients with hypercoagulable states.
AUTHOR: Watanabe R; Wada H; Sakakura M; Mori Y; Nakasaki T; Okugawa Y; Gabazza E C; Hayashi T; Nishioka J; Suzuki K; Shiku H; Nobori T
CORPORATE SOURCE: Second Department of Internal Medicine, Mie University School of Medicine, Tsu-city, Mie-ken, Japan.
SOURCE: AMERICAN JOURNAL OF HEMATOLOGY, (2000 Sep) 65 (1) 35-40. Journal code: 3H4; 7610369. ISSN: 0361-8609.
PUB. COUNTRY: United States
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20000915
Last Updated on STN: 20000915
Entered Medline: 20000907

AB Plasma levels of activated **protein C** (APC)-**protein C** inhibitor (PCI) were significantly increased in patients with disseminated intravascular coagulation (DIC), thrombotic **thrombocytopenic purpura** (TTP), acute myocardial infarction (AMI), pulmonary embolism (PE), or deep vein thrombosis (DVT) and in patients undergoing hemodialysis (HD). Plasma levels of APC-alpha(1)-antitrypsin (AT) complex were significantly increased in patients with DIC and in those with TTP. Plasma levels of PCI were significantly decreased in patients with DIC, non-DIC, or TTP and in those

undergoing HD. In the pre-DIC stage, the plasma levels of APC-PCI complex were significantly increased but not those of APC-alpha(1)-AT complex. These data suggest that measurements of APC-PCI complex and APC-alpha(1)-AT complex may be useful for the diagnosis of DIC. After **treatment** of DIC, the plasma levels of APC-PCI complex and APC-alpha(1)-AT complex were significantly decreased, but not those of PCI. Plasma levels of thrombin-antithrombin complex (TAT), plasmin-alpha(2)-plasmin complex (PPIC), D-dimer, and soluble fibrin monomer (SFM) were markedly increased in patients with DIC or pre-DIC and were moderately increased in patients with non-DIC, TTP, AMI, PE, or DVT

and in those undergoing HD. The receiving operating characteristic (ROC) analysis showed that SFM and the APC-PCT complex are useful markers for diagnosis of DIC. The specificity of plasma TAT and PPIC levels was low. The positive rate of APC-PCI complex was higher than 90% with DIC, TTP, AMI, PE, and it was higher than 60% with DVT and HD. Since the APC-PCI complex was elevated not only in patients with venous thrombosis but also in those with arterial thrombosis, components of the **protein C** pathway might be useful markers for the diagnosis of arterial thrombosis.

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L8 ANSWER 9 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 1999030394 EMBASE

TITLE: Acute generalized, widespread bleeding. Diagnosis and management.

AUTHOR: Rocha E.; Paramo J.A.; Montes R.; Panizo C.

CORPORATE SOURCE: Dr. E. Rocha, Hematology Service, Clinica Universitaria, Universidad de Navarra, Pamplona, Spain. erocha@unav.es

SOURCE: Haematologica, (1998) 83/11 (1024-1037).

Refs: 194

ISSN: 0390-6078 CODEN: HAEMAX

COUNTRY: Italy

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 025 Hematology

037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Background and Objective. Acute generalized, widespread bleeding is often related to disseminated intravascular coagulation (DIC), a pathologic process which complicates the clinical course of many diseases and is characterized by huge amounts of thrombin and plasmin within the circulation. The final result is the consumption of platelets, coagulation

factors and inhibitors, as well as secondary hyperfibrinolysis, all leading to diffuse hemorrhage and microthromboses. This review article examines the present attitudes to the diagnosis and **treatment** of overt DIC in clinical practice, emphasizing the importance of an accurate differential diagnosis from some other processes characterized by acute generalized, widespread bleeding. Information Sources. The authors have been working in this field, both at experimental and clinical levels, contributing original papers for many years, in addition, material examined in this review includes articles published in journals covered

by

MedLine, recent reviews in journals with high impact factor and in relevant books on hemostasis and thrombosis. State of Art and Perspectives. DIC is an intermediary mechanism of disease which complicates the clinical course of many well-known disorders. Although

the

systemic hemorrhagic syndrome is the predominant clinical manifestation, massive intravascular thrombosis frequently occurs contributing to ischemia and associated organ damage, making the mortality rate of this condition high. Current concepts on the pathophysiology, laboratory diagnosis and management of DIC are presented. Complex pathophysiological interrelations make the diagnosis of the etiology of the DIC difficult in clinical practice, although simple tests are useful for identification of patients with the process. Laboratory diagnosis of DIC is mainly based on screening assays, which allow a rapid diagnosis, whereas some other

highly

sensitive but more complex assays are not always available to routine clinical laboratories. The management of DIC is based on the **treatment** of the underlying disease, supportive and replacement therapies and the control of the coagulation mechanisms. Although some advances have been achieved, management decisions are still

controversial,

so that therapy should be highly individualized depending on the nature

of

the DIC and severity of clinical symptoms. Many syndromes sharing common findings with DIC such as primary hyperfibrinolysis or thrombotic thrombocytopenic purpura, should be excluded. Finally, new therapeutic approaches to the management of this potentially catastrophic syndrome are required.

L8 ANSWER 10 OF 16 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:840527 SCISEARCH
THE GENUINE ARTICLE: 133GE
TITLE: Thrombomodulin: an overview and potential implications in vascular disorders
AUTHOR: Boffa M C (Reprint); Karmochkine M
CORPORATE SOURCE: HOP ST LOUIS, INSERM, U353, INST HEMATOL, F-75475 PARIS 10, FRANCE (Reprint); HOP BROUSSAIS, SERV IMMUNOL CLIN, F-75674 PARIS, FRANCE
COUNTRY OF AUTHOR: FRANCE
SOURCE: LUPUS, (WIN 1998) Vol. 7, Supp. [2], pp. S120-S125.
Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE RG21 6XS, HAMPSHIRE, ENGLAND.
ISSN: 0961-2033.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: CLIN
LANGUAGE: English
REFERENCE COUNT: 68

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Thrombomodulin (TM), a high affinity thrombin receptor present on endothelial cell membrane, plays an important role as a natural anticoagulant. It acts as a cofactor of thrombin-catalyzed activation of protein C, and inhibits the procoagulant functions of thrombin. TM is also located in other cells (keratinocytes, osteoblasts, macrophages,...) where it might be involved in cell differentiation or in inflammation. In the presence of cytokines, activated neutrophils and macrophages, endothelial TM is cleaved enzymatically, releasing soluble fragments which circulate in the blood and are eliminated in urine.

Plasma

TM level (pTM) can be measured using a two-site enzyme-linked immunosorbent assay (ELISA). pTM level is regarded as a molecular marker reflecting injury of endothelial cells. It is often increased in case of diffuse endothelial damage as in disseminated intravascular coagulation, diabetic microangiopathy, Plasmodium falciparum and rickettsial infections, pTM is also a predictive marker of hypertensive complications in pregnancy. In several systemic inflammatory diseases, pTM levels are correlated to the activity of the disease.

L8 ANSWER 11 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97041938 EMBASE
DOCUMENT NUMBER: 1997041938
TITLE: Blood and blood product transfusion, indications, and proper use.
AUTHOR: Dzieczkowski J.
CORPORATE SOURCE: Dr. J. Dzieczkowski, Hutzel Hospital-Blood Bank, 4707 St. Antoine Boulevard, Detroit, MI 48201, United States
SOURCE: Infertility and Reproductive Medicine Clinics of North America, (1997) 8/1 (109-124).
Refs: 67
ISSN: 1047-9422 CODEN: IRMCF8
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 010 Obstetrics and Gynecology
025 Hematology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Indications for the use of blood components have been under close scrutiny since the increased fear of infectious complications. A review of transfusion practices reveals that many once accepted criteria were

unwarranted and have subsequently been modified. The currently accepted criteria for the proper use of various blood products are summarized as follows: Packed red blood cells: Hemoglobin level < 7 g/dL in a patient with normal cardiovascular function; Higher levels of hemoglobin are acceptable for transfusion if the patient has complicating factors such

as

cardiovascular disease, hemoglobinopathy, sepsis, or critical illness; Acceptable indications for autologous transfusion vary with institutional policy. Platelets: Prophylactically for platelet counts $< 10,000/\text{mm}^3$; With a platelet count $< 20,000/\text{mm}^3$ and bleeding or a minor procedure; With a platelet count $< 50,000/\text{mm}^3$ in a patient scheduled for a major procedure; Documented dysfunctional platelets with bleeding or scheduled procedure; Contraindicated in immune **thrombocytopenic purpura**. Fresh-frozen plasma: Multiple coagulation factor deficiencies with prothrombin time or partial thromboplastin time or both > 1.5 normal; Deficiencies of factors II, V, VII, X, XII, XIII or **proteins C or S**; Emergency reversal of oral anticoagulant. Cryoprecipitate: **Treatment** for: Hemophilia A, when concentrates not available von Willebrand's disease; Fibrinogen < 100 mg/dL; Dysfibrinogenemia. Production of fibrin glue. Clinicians who may encounter situations in which transfusion therapy is required are wise to keep abreast of current recommendations.

L8 ANSWER 12 OF 16 MEDLINE

ACCESSION NUMBER: 97187108 MEDLINE

DOCUMENT NUMBER: 97187108 PubMed ID: 9034561

TITLE: Plasma levels of activated FVII in various diseases.

AUTHOR: Yamada A; Wada H; Kamikura Y; Hiroyama K; Shimura M; Nagaya

CORPORATE SOURCE: S; Deguchi K; Mori Y; Shiku H
2nd Department of Internal Medicine, Mie University School of Medicine, Japan.

SOURCE: BLOOD COAGULATION AND FIBRINOLYSIS, (1996 Nov) 7 (8)
794-8.

Journal code: A5J; 9102551. ISSN: 0957-5235.

PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199704

ENTRY DATE: Entered STN: 19970507

Last Updated on STN: 19970507

Entered Medline: 19970429

AB Plasma activated factor VIIa (FVIIa) levels were measured in various diseases using mutant tissue factor (TF). FVIIa levels in thrombotic patients and patients with idiopathic **thrombocytopenic purpura** were significantly higher than those in healthy control subjects. The plasma FVIIa levels in thrombotic patients **treated** with warfarin were similar to those of control subjects. The plasma FVIIa levels in pregnant women and patients with systemic lupus erythematosus, infection or malignancies were high. However, the levels in patients with disseminated intravascular coagulation (DIC) were not significantly increased. DIC patients are in a severe hypercoagulable state, and

exhibit

severe consumption of coagulation factors. The slightly increased FVIIa level in the DIC patients observed is probably considered to be caused by consumption of coagulation factors. The plasma FVIIa level was poorly correlated with other hemostatic parameters except for **protein C** in our analysis of all cases. In the analysis of DIC and thrombotic patients **treated** without warfarin, the plasma FVIIa level was negatively correlated with TF antigen. Plasma FVIIa levels

might

reflect hypercoagulability in thrombotic diseases, and a normalized FVIIa level in patients with thrombotic diseases should be considered to be associated with DIC.

L8 ANSWER 13 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 96700796 EMBASE
DOCUMENT NUMBER: 199000796
TITLE: Coagulation disorders in cancer.
AUTHOR: Goad K.E.; Gralnick H.R.
CORPORATE SOURCE: National Institutes of Health, Building 10, 9000 Rockville Pike, Bethesda, MD 20892, United States
SOURCE: Hematology/Oncology Clinics of North America, (1996) 10/2 (457-484).
ISSN: 0889-8588 CODEN: HCNAEQ
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
018 Cardiovascular Diseases and Cardiovascular Surgery
025 Hematology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Coagulation disorders are common in cancer patients. This article reviews the coagulation laboratory findings in these patients and the thromboembolic and hemorrhagic manifestations of malignancy. Among the many topics addressed are Trousseau's syndrome, disseminated intravascular coagulation, and acquired von Willebrand disease. Pathogenesis of the coagulation disorders and recommendations for treatment of various syndromes are discussed.

L8 ANSWER 14 OF 16 MEDLINE
ACCESSION NUMBER: 97104572 MEDLINE
DOCUMENT NUMBER: 97104572 PubMed ID: 9005011
TITLE: [Disseminated intravascular coagulations].
Les coagulations intra-vasculaires disseminees.
AUTHOR: Amstutz P; Moyo J S
CORPORATE SOURCE: Service de Reanimation, Hopital Saint-Antoine, Paris.
SOURCE: CAHIERS D ANESTHESIOLOGIE, (1996) 44 (3) 219-28. Ref: 59
Journal code: CBV; 0370650. ISSN: 0007-7625.
PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19970219
Entered Medline: 19970128

AB Disseminated intravascular coagulation (DIC) syndromes can be defined as the formation of fibrin deposits within the microcirculation, occurring in definite clinical situations. Their biological counterpart is a consumption coagulopathy. The clinical profiles of DIC have been well known for decades, are multiform and range from latency to overwhelming haemorrhagic diatheses, including also characteristic but rare situations, such as purpura fulminans, acral cyanosis and pictures resembling thrombotic thrombocytopenic purpura or haemolytic-uraemic syndrome. Biological tests of DIC show a consumption coagulopathy, displayed on the standard haemostasis sheet; along with signs of paracoagulation and/or of secondary fibrinolysis (FDP). New tests

have recently been introduced: D-dimers are specific and sensible; Antithrombin-III, protein C and alpha 2-antiplasmin also can sometimes be useful. The knowledge of the pathophysiology of DIC has made advances with passing years. Fibrin deposits may be non-occlusive, and indeed they are swiftly removed by a secondary

fibrinolysis. Except in very rare situations, such as those leading to a cortical renal necrosis, and perhaps in some ARDS, there is little evidence relating DIC to organ failure syndromes. Moreover, there is no clear relationship between the severity of the consumption coagulopathy and the prognosis. For instance, the mortality is much lower in abruptio placentae, where the coagulopathy is very severe, than in septic shock, where it is usually moderate. In septic shock, the disorders of haemostasis were related initially to a platelet activation, then to an activation of the contact system (releasing kinins and triggering complement cascade), and nowadays to the activation of the extrinsic coagulation system. The **treatment** of DIC is mainly the **treatment** of its cause. Indications for heparin therapy should be strictly limited to a few exceptional circumstances. When haemorrhagic diathesis threatens, FPC and/or platelet transfusion may be indicated. Aprotinin can be useful in rare cases of overwhelming secondary fibrinolysis. Trials with antithrombin-III or C1-esterase inhibitors are in progress.

L8 ANSWER 15 OF 16 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 93:460692 SCISEARCH
THE GENUINE ARTICLE: LN492
TITLE: ETIOLOGY OF STROKE IN CHILDREN
AUTHOR: RIELA A R (Reprint); ROACH E S
CORPORATE SOURCE: UNIV TEXAS, SW MED CTR, DEPT NEUROL, DIV PEDIAT NEUROL,
5323 HARRY HINES BLVD, DALLAS, TX, 75235 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CHILD NEUROLOGY, (JUL 1993) Vol. 8, No. 3, pp.
201-220.
ISSN: 0883-0738.
DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 126

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Cerebrovascular disorders are more common than once suspected, and our ability to diagnose stroke in children has improved with the development of newer imaging techniques in recent years. Children have a wide array of risk factors that promote cerebral infarction or hemorrhage, and a likely cause can eventually be pinpointed in about two thirds of patients if a thorough diagnostic evaluation is performed. Ideally, a systematic evaluation should confirm the presence of a cerebrovascular lesion and also identify the cause, concentrating initially on the more common or **treatable** risk factors. Recognition of the cause of a child's stroke is important, because the likelihood of recurrence depends largely on the etiology and whether **treatment** is available.

L8 ANSWER 16 OF 16 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 94:33454 SCISEARCH
THE GENUINE ARTICLE: MP089
TITLE: HEMATOLOGICAL MANIFESTATIONS OF SYSTEMIC LUPUS-ERYTHEMATOSUS
AUTHOR: KEELING D M (Reprint); ISENBERG D A
CORPORATE SOURCE: ADDENBROOKES HOSP, DEPT HAEMATOL, HILLS RD, CAMBRIDGE CB2 2QQ, ENGLAND (Reprint); UNIV COLL LONDON, BLOOSBURY RHEUMATOL UNIT, LONDON W1P 9PG, ENGLAND
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ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Haematological involvement is common in systemic lupus erythematosus (SLE). Whilst anaemia is most often due to chronic disease, other causes

such as autoimmune haemolytic anaemia and hypoplastic anaemia need to be considered. The increased risk of infection in patients with SLE is due

in

part to changes in the white blood cells though **treatments** do not yet aim to modify these. Thrombocytopenia occurs frequently and is almost invariably autoimmune. It is often of little consequence, but may occasionally be severe and serious, requiring aggressive **treatment**. Patients with SLE have an increased risk of thrombosis, increased further in the presence of antiphospholipid antibodies (aPL). Changes in the haemostatic system and new insights into the nature of aPL are